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L44 ANSWER 1 OF 7 MEDLINE on STN
AN 2005246876 MEDLINE
DN PubMed ID: 15887106
TI Recombinant probiotics for treatment and prevention of
enterotoxigenic Escherichia coli
diarrhea.
AU Paton Adrienne W; Jennings Michael P; Morona Renato; Wang Hui; Focareta
Antonio; Roddam Louise F; Paton James C
CS School of Molecular and Biomedical Science, University of Adelaide, South
Australia, Australia.
SO Gastroenterology, (2005 May) Vol. 128, No. 5, pp. 1219-28.
Journal code: 0374630. ISSN: 0016-5085.
CM Comment in: Gastroenterology. 2005 May;128(5):1509-12. PubMed ID: 15887131
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 200507
ED Entered STN: 12 May 2005
Last Updated on STN: 6 Jul 2005
Entered Medline: 5 Jul 2005
AB BACKGROUND & AIMS: We have developed a therapeutic strategy for
gastrointestinal infections that is based on molecular mimicry of host
receptors for bacterial toxins on the surface of harmless gut bacteria.
The aim of this study was to apply this to the development of a
recombinant probiotic for treatment and prevention of **diarrheal**
disease caused by **enterotoxigenic Escherichia**
coli strains that produce heat-labile **enterotoxin**.
METHODS: This was achieved by expressing glycosyltransferase genes from
Neisseria meningitidis or *Campylobacter jejuni* in a harmless
Escherichia coli strain (CWG308), resulting in the
production of a chimeric lipopolysaccharide capable of binding heat-labile
enterotoxin with high avidity. RESULTS: The strongest heat-labile
enterotoxin binding was achieved with a construct (CWG308:pLNT)
that expresses a mimic of **lacto-N-neotetraose**, which
neutralized > or = 93.8% of the heat-labile **enterotoxin** activity
in culture lysates of diverse **enterotoxigenic**
Escherichia coli strains of both human and porcine
origin. When tested with purified heat-labile **enterotoxin**, it
was capable of adsorbing approximately 5% of its own weight of toxin.
Weaker toxin neutralization was achieved with a construct that mimicked
the ganglioside GM2. Preabsorption with, or coadministration of,
CWG308:pLNT also resulted in significant in vivo protection from
heat-labile **enterotoxin**-induced fluid secretion in rabbit
ligated ileal loops. CONCLUSIONS: Toxin-binding probiotics such as those
described here have considerable potential for prophylaxis and treatment
of **enterotoxigenic Escherichia coli**-induced

travelers' **diarrhea**.

CT Adrenal Glands: CY, cytology
 Animals
 Bacterial Toxins: ME, metabolism
 Campylobacter jejuni: GE, genetics
 Cells, Cultured
 Cholera Toxin: ME, metabolism
 *Diarrhea: MI, microbiology
 *Diarrhea: PC, prevention & control
 Enterotoxins: ME, metabolism
 *Escherichia coli: CL, classification
 *Escherichia coli: GE, genetics
 Escherichia coli: ME, metabolism
 Glycosyltransferases: GE, genetics
 Ileum: MI, microbiology
 Lipopolysaccharides: ME, metabolism
 Neisseria meningitidis: GE, genetics
 *Probiotics: PD, pharmacology
 Rabbits
 Recombinant Proteins: GE, genetics

RN 9012-63-9 (Cholera Toxin)

CN 0 (Bacterial Toxins); 0 (Enterotoxins); 0 (Lipopolysaccharides);
 0 (Recombinant Proteins); 0 (heat stable toxin (**E coli**
)); EC 2.4.- (Glycosyltransferases)

L44 ANSWER 2 OF 7 MEDLINE on STN

AN 2001060702 MEDLINE

DN PubMed ID: 10965049

TI Common architecture of the primary galactose binding sites of Erythrina
 corallodendron lectin and heat-labile **enterotoxin** from
Escherichia coli in relation to the binding of branched
neolactohexaosylceramide.

AU Teneberg S; Berntsson A; Angstrom J

CS Institute of Medical Biochemistry, Goteborg University, P.O. Box SE 405 30
 Goteborg, Sweden.

SO Journal of biochemistry, (2000 Sep) Vol. 128, No. 3, pp. 481-91.
 Journal code: 0376600. ISSN: 0021-924X.

CY Japan

DT (COMPARATIVE STUDY)
 Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)

LA English

FS Priority Journals

EM 200012

ED Entered STN: 22 Mar 2001
 Last Updated on STN: 18 Dec 2002
 Entered Medline: 22 Dec 2000

AB The heat-labile **enterotoxin** from **Escherichia coli** (LT) is responsible for so-called traveller's **diarrhea** and is closely related to the cholera toxin (CT). Toxin binding to GM1 at the epithelial cell surface of the small intestine initiates the subsequent **diarrheal** disease. However, LT has a broader receptor specificity than CT in that it also binds to N-acetyllactosamine-terminated structures. The unrelated lectin from Erythrina corallodendron (ECorL) shares this latter binding property. The findings that both ECorL and porcine LT (pLT) bind to lactose as well as to **neolactotetraosylceramide** suggests a common structural theme in their respective primary binding sites. Superimposing the terminal galactose of the lactoses in the respective crystal structures of pLT and ECorL reveals striking structural similarities around the galactose

despite the lack of sequence and folding homology, whereas the interactions of the penultimate GlcNAcb3 in the **neolactotetraosylceramide** differ. The binding of branched **neolactohexaosylceramide** to either protein reveals an enhanced affinity relative to **neolactotetraosylceramide**. The b3-linked branch is found to bind to the primary Gal binding pocket of both proteins, whereas the b6-linked branch outside this site provides additional interactions in accordance with the higher binding affinities found for this compound. While the remarkable architectural similarities of the primary galactose binding sites of pLT and ECorL point to a convergent evolution of these subsites, the distinguishing structural features determining the overall carbohydrate specificities are located in extended binding site regions. In pLT, Arg13 is thus found to play a crucial role in enhancing the affinity not only for N-acetyllactosamine-terminated structures but also for GM1 as compared to human LT (hLT) and CT. The physiological relevance of the binding of N-acetyllactosamine-containing glycoconjugates to LT and ECorL is briefly discussed.

CT Amino Sugars: ME, metabolism
Animals
*Antigens, CD
*Bacterial Toxins: ME, metabolism
Crystallography, X-Ray
*Enterotoxins: ME, metabolism
*Erythrina: ME, metabolism
*Escherichia coli: ME, metabolism
*Escherichia coli Proteins
*Galactose: ME, metabolism
Humans
Hydrogen Bonding
Isotope Labeling
*Lactosylceramides: ME, metabolism
*Lectins: ME, metabolism
Ligands
Magnetic Resonance Spectroscopy
Models, Molecular
Plant Lectins
*Plants, Medicinal
Protein Conformation
Structure-Activity Relationship
Swine

RN 26566-61-0 (Galactose); 32181-59-2 (N-acetyllactosamine); 4682-48-8 (CDw17 antigen)

CN 0 (Amino Sugars); 0 (Antigens, CD); 0 (Bacterial Toxins); 0 (Enterotoxins); 0 (Escherichia coli Proteins); 0 (Lactosylceramides); 0 (Lectins); 0 (Ligands); 0 (Plant Lectins); 0 (enterotoxin LT)

L44 ANSWER 3 OF 7 MEDLINE on STN

AN 97403073 MEDLINE

DN PubMed ID: 9258442

TI Immobilization of reducing sugars as toxin binding agents.

AU Nilsson U J; Heerze L D; Liu Y C; Armstrong G D; Palcic M M; Hindsgaul O

CS Department of Chemistry, University of Alberta, Edmonton, Canada.

SO Bioconjugate chemistry, (1997 Jul-Aug) Vol. 8, No. 4, pp. 466-71.

Journal code: 9010319. ISSN: 1043-1802.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

(RESEARCH SUPPORT, NON-U.S. GOV'T)

LA English

FS Priority Journals
EM 199710
ED Entered STN: 24 Oct 1997
Last Updated on STN: 24 Oct 1997
Entered Medline: 10 Oct 1997
AB A simple and economical procedure for the attachment of reducing sugars to aminated solid supports has been developed. Reaction of the amino groups on the solid support with p-nitrophenyl chloroformate, followed by 1,6-hexanediamine, yields a chain-extended amine to which reducing sugars can be attached while remaining accessible to macromolecules. Immobilization of the reducing sugars involves a simple incubation followed by trapping of the resulting glycosylamine with acetic anhydride and recovery of the unreacted sugar by filtration. This technique was used to immobilize lactose and **sialyllactose** onto silylaminated Chromosorb P, producing solid supports that effectively neutralized the activity of cholera toxin from *Vibrio cholerae* and heat-labile **enterotoxin of enterotoxigenic Escherichia coli**. The general applicability of such solid supports for toxin neutralization was further demonstrated by immobilization of the enzymatically synthesized alpha Gal(1-3) beta Gal(1-4)Glc trisaccharide, which produced a support that efficiently neutralized toxin A of *Clostridium difficile*. The results from this study suggest that these solid supports have the potential to serve as inexpensive therapeutics for bacterial toxin-mediated **diarrheal** diseases.
CT Animals
*Bacterial Toxins: ME, metabolism
CHO Cells
Carbohydrate Sequence
*Cholera Toxin: ME, metabolism
Cricetinae
*Enterotoxins: ME, metabolism
Escherichia coli: CH, chemistry
*Escherichia coli Proteins
Molecular Sequence Data
*Oligosaccharides: CH, chemistry
Oligosaccharides: ME, metabolism
Oxidation-Reduction
Protein Binding
RN 9012-63-9 (Cholera Toxin)
CN 0 (Bacterial Toxins); 0 (Enterotoxins); 0 (Escherichia coli Proteins); 0 (Oligosaccharides); 0 (enterotoxin LT); 0 (tcdA protein, Clostridium difficile)
L44 ANSWER 4 OF 7 MEDLINE on STN
AN 95252586 MEDLINE
DN PubMed ID: 7766178
TI Inhibition of cholera toxin by human milk fractions and **sialyllactose**.
AU Idota T; Kawakami H; Murakami Y; Sugawara M
CS Technical Research Institute, Snow Brand Milk Products Co., Ltd., Saitama, Japan.
SO Bioscience, biotechnology, and biochemistry, (1995 Mar) Vol. 59, No. 3, pp. 417-9.
Journal code: 9205717. ISSN: 0916-8451.
CY Japan
DT (IN VITRO)
Journal; Article; (JOURNAL ARTICLE)
LA English
FS Biotechnology
EM 199506

ED Entered STN: 9 Aug 1995
 Last Updated on STN: 9 Aug 1995
 Entered Medline: 8 Jun 1995

AB The effects of human milk fractions on cholera toxin B subunit binding to monosialoganglioside 1 (GM1) were investigated. Human milk, human defatted milk, whey, and a low-molecular-weight fraction of human milk inhibited the binding, but casein did not inhibit it. The inhibitory activity of whey from bovine-milk-based infant formula was less than that of whey from human milk. Differences in composition between human and bovine whey seemed to influence the extent of the inhibitory activity. Sialylated oligosaccharides were considered to be the possible components that inhibited cholera toxin. The effects of **sialyllactose**, a predominant sialylated component of human milk, on cholera toxin-induced **diarrhea** were investigated by the rabbit intestinal loop method. **Sialyllactose** inhibited the cholera toxin inducing fluid accumulation, although neither sialic acid nor lactose had an effect on it. The results suggest that **sialyllactose** is responsible for the inhibitory activity of milk on cholera toxin.

CT Check Tags: Male
 Animals
 Binding, Competitive: DE, drug effects
 Body Fluids: DE, drug effects
 *Cholera Toxin: AI, antagonists & inhibitors
 G(M1) Ganglioside: PD, pharmacology
 Humans
 Intestines: DE, drug effects
 Intestines: ME, metabolism
 *Lactose: AA, analogs & derivatives
 Lactose: PD, pharmacology
 *Milk, Human: CH, chemistry
 Oligosaccharides: IP, isolation & purification
 Oligosaccharides: PD, pharmacology
 Rabbits
 *Sialic Acids: PD, pharmacology

RN **35890-38-1 (N-acetylneuraminoyllactose)**; 37758-47-7 (G(M1) Ganglioside); 63-42-3 (Lactose); 9012-63-9 (Cholera Toxin)

CN 0 (Oligosaccharides); 0 (Sialic Acids)

L44 ANSWER 5 OF 7 MEDLINE on STN
 AN 93293800 MEDLINE
 DN PubMed ID: 8514738
 TI Postnatal change of pig intestinal ganglioside bound by **Escherichia coli** with K99 fimbriae.
 AU Yuyama Y; Yoshimatsu K; Ono E; Saito M; Naiki M
 CS Faculty of Veterinary Medicine, Hokkaido University.
 SO Journal of biochemistry, (1993 Apr) Vol. 113, No. 4, pp. 488-92.
 Journal code: 0376600. ISSN: 0021-924X.
 CY Japan
 DT Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 LA English
 FS Priority Journals
 EM 199307
 ED Entered STN: 6 Aug 1993
 Last Updated on STN: 6 Aug 1993
 Entered Medline: 22 Jul 1993

AB **Enterotoxigenic Escherichia coli** possessing K99 fimbriae (**E. coli** K99) causes **diarrhea** in piglets of less than 1 week old. The first stage of the bacterial infection is adhesion by the fimbriae on the small intestinal mucosa and

the adhesion is followed by colony formation. K99 fimbriae bind specifically to **N-glycolylneuraminyl-lactosyl-ceramide**, GM3(NeuGc) [Ono, E. et al. (1989) Infect. Immun. 57,907-911]. We examined the postnatal change of the content and the molecular species of GM3(NeuGc) in the small intestinal mucosa of 0- to 14-day-old piglets and adult pigs. GM3(NeuGc) was a major ganglioside of piglet intestinal mucosa. GM3(NeuGc) content was maximal at birth and gradually decreased to 1/16 in adult animals (5 months old). The **ceramide** moiety of piglet intestinal GM3(NeuGc) was characterized by the presence of 2-hydroxylated palmitic acid. 125I-labeled bacteria strongly bound to GM3(NeuGc) containing 2-hydroxylated palmitic acid and phytosphingosine compared with GM3(NeuGc) containing any other **ceramide** moiety. The time when this particular GM3(NeuGc) appears coincides with the time that the infection occurs, and it may explain the susceptibility of newborn piglets to *E. coli* K99 infection.

CT Animals
Animals, Newborn
Chromatography, Thin Layer
Enterotoxins
**Escherichia coli*: PY, pathogenicity
**Escherichia coli* Infections: MI, microbiology
*G(M3) Ganglioside: AA, analogs & derivatives
G(M3) Ganglioside: ME, metabolism
Gangliosides: ME, metabolism
*Intestinal Diseases: MI, microbiology
*Intestinal Mucosa: ME, metabolism
*Intestinal Mucosa: MI, microbiology
Intestine, Small: ME, metabolism
Intestine, Small: MI, microbiology
Swine
RN 69345-49-9 (N-glycolylneuraminyl-lactosylceramide)
CN 0 (Enterotoxins); 0 (G(M3) Ganglioside); 0 (Gangliosides)

L44 ANSWER 6 OF 7 MEDLINE on STN
AN 89339746 MEDLINE
DN PubMed ID: 2503449
TI Hemagglutinating properties of *Shigella dysenteriae* type 1 and other *Shigella* species.
AU Qadri F; Haq S; Ciznar I
CS Laboratory Sciences Division, International Centre for Diarrhoeal Disease and Research, Bangladesh.
SO Infection and immunity, (1989 Sep) Vol. 57, No. 9, pp. 2909-11.
Journal code: 0246127. ISSN: 0019-9567.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198909
ED Entered STN: 9 Mar 1990
Last Updated on STN: 9 Mar 1990
Entered Medline: 15 Sep 1989
AB Strains of *Shigella dysenteriae* type 1 cultured in Casamino Acids-yeast extract broth medium in the presence of 1 mM calcium chloride at 37 degrees C for 22 h induced hemagglutination of erythrocytes that was inhibited by N-acetylneuraminic acid, N-acetylneuramin-lactose, and alpha 1-glycoprotein. The hemagglutination was heat labile, and the absence of cell-surface appendages suggested a nonfimbrial adhesin(s). Under the same conditions, strains of *Shigella flexneri* (types 1a, 1b, 2a, and 2b) showed N-acetylneuraminic

acid-resistant hemagglutination of erythrocytes.

CT Animals
 Cattle
 Culture Media: AN, analysis
 Guinea Pigs
 Haplorhini
 *Hemagglutination Tests
 Humans
 Rabbits
 Sheep
 Shigella dysenteriae: GD, growth & development
 ***Shigella dysenteriae: IM, immunology**
 Swine

CN 0 (Culture Media)

L44 ANSWER 7 OF 7 MEDLINE on STN
 AN 79216779 MEDLINE
 DN PubMed ID: 222809
 TI Gangliosides sensitize unresponsive fibroblasts to **Escherichia coli** heat-labile **enterotoxin**.
 AU Moss J; Garrison S; Fishman P H; Richardson S H
 SO The Journal of clinical investigation, (1979 Aug) Vol. 64, No. 2, pp. 381-4.
 Journal code: 7802877. ISSN: 0021-9738.
 CY United States
 DT Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
 LA English
 FS Abridged Index Medicus Journals; Priority Journals
 EM 197909
 ED Entered STN: 15 Mar 1990
 Last Updated on STN: 15 Mar 1990
 Entered Medline: 25 Sep 1979

AB Chemically transformed mouse fibroblasts did not raise their cyclic AMP level in response to **Escherichia coli** heat-labile **enterotoxin**. These fibroblasts did, however, incorporate exogenous mono-, di-, and trisialogangliosides. After the uptake of monosialoganglioside galactosyl-N-acetylgalactosaminyl-[N-acetylneuraminy]-galactosylglucosylceramide (GM1), the cells responded to **E. coli** heat-labile **enterotoxin**. The di- and trisialogangliosides were considerably less effective. GM1, the putative cholera toxin (cholera toxin) receptor, has been implicated previously as the receptor for **E. coli** heat-labile **enterotoxin** based on the ability of the free ganglioside to inhibit the effects of toxin. This investigation establishes that the ganglioside, when incorporated into fibroblasts, serves a functional role in mediating the responsiveness to the toxin.

CT Animals
 Cell Line
 Cholera Toxin: PD, pharmacology
 *Cyclic AMP: ME, metabolism
 ***Enterotoxins: PD, pharmacology**
 ***Escherichia coli**
 Fibroblasts: DE, drug effects
 *Fibroblasts: ME, metabolism
 *Gangliosides: PD, pharmacology
 Heat
 Mice

RN 60-92-4 (Cyclic AMP); 9012-63-9 (Cholera Toxin)
 CN 0 (Enterotoxins); 0 (Gangliosides)

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L58 ANSWER 1 OF 16 MEDLINE on STN
AN 2007131051 MEDLINE
DN PubMed ID: 17268859
TI Conjugation of oligosaccharides by reductive amination to amine modified chondroitin oligomer and gamma-cyclodextrin.
AU Weikkolainen Krista; Aitio Olli; Blomqvist Maria; **Natunen Jari**; Helin Jari
CS Department of Biological and Environmental Sciences, University of Helsinki, P. O. Box 56, 00014, Helsinki, Finland.
SO Glycoconjugate journal, (2007 Apr) Vol. 24, No. 2-3, pp. 157-65.
Electronic Publication: 2007-02-01.
Journal code: 8603310. ISSN: 0282-0080.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 200711
ED Entered STN: 3 Mar 2007
Last Updated on STN: 9 Nov 2007
Entered Medline: 8 Nov 2007
AB Carbohydrates present on cell surfaces participate in numerous biological recognition phenomena including cell-cell interactions, cancer metastasis and pathogen invasion. Therefore, synthetic carbohydrates have a potential to act as pharmaceutical substances for treatment of various pathological phenomena by inhibiting specifically the interaction between cell surface carbohydrates and their protein receptors (lectins). However, the inherently low affinity of carbohydrate-protein interactions has often been an obstacle for successful generation of carbohydrate based pharmaceuticals. Multivalent glycoconjugates, i.e. structures carrying several copies of the active carbohydrate sequence in a carrier molecule, have been constructed to overcome this problem. Here we present two novel types of multivalent carbohydrate conjugates based on chondroitin oligomer and cyclodextrin carriers. These carriers were modified to express primary amino groups, and oligosaccharides were then bound to carrier molecules by reductive amination. Multivalent conjugates were produced using the human milk type oligosaccharides LNDFH I (Lewis-b hexasaccharide), **LNnT**, and GlcNAcbetal-3Galbetal-4GlcNAcbetal-3Galbetal-4Glc.

L58 ANSWER 2 OF 16 MEDLINE on STN
AN 2006599489 MEDLINE
DN PubMed ID: 16880000
TI Binding of Haemophilus ducreyi to carbohydrate receptors is mediated by the 58.5-kDa GroEL heat shock protein.
AU Pantzar Martina; **Teneberg Susann**; Lagergard Teresa
CS Institute of Biomedicine, Department of Microbiology and Immunology, Goteborg University, PO Box 435, SE-40530, Goteborg, Sweden.
SO Microbes and infection / Institut Pasteur, (2006 Aug) Vol. 8, No. 9-10, pp. 2452-8. Electronic Publication: 2006-07-07.
Journal code: 100883508. ISSN: 1286-4579.
CY France
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals

EM 200702
ED Entered STN: 11 Oct 2006
Last Updated on STN: 14 Feb 2007
Entered Medline: 13 Feb 2007

AB The bacterium *Haemophilus ducreyi* causes the sexually transmitted disease chancroid, which is characterized by the appearance of mucocutaneous, persistent ulcers on the external genitals. To identify carbohydrate receptors that mediate the attachment of this pathogen to host cells, we investigated the binding of 35S-methionine-labeled *H. ducreyi* strains to a panel of defined glycosphingolipids that were separated on thin layer chromatography plates. *H. ducreyi* bound to lactosylceramide, gangliotriaosylceramide, gangliotetraosylceramide, **neolactotetraosylceramide**, the GM3 ganglioside, and sulfatide. To elucidate the role of the surface-located 58.5-kDa GroEL heat shock protein (HSP) of *H. ducreyi* in attachment, we investigated the binding of purified HSP to the same panel of glycosphingolipids. Our results suggest that the 58.5-kDa GroEL HSP of *H. ducreyi* is responsible for the attachment of this bacterium to the majority of the tested glycosphingolipids, and thus represents a potential bacterial adhesin.

L58 ANSWER 3 OF 16 MEDLINE on STN
AN 2006283542 MEDLINE
DN PubMed ID: 16714580
TI The major subunit, CfaB, of colonization factor antigen i from **enterotoxigenic Escherichia coli** is a glycosphingolipid binding protein.
AU Jansson Lena; Tobias Joshua; Lebens Michael; Svennerholm Ann-Mari; **Teneberg Susann**
CS Department of Medical Biochemistry, Institute of Biomedicine, Goteborg University, P.O. Box 440, S-405 30 Goteborg, Sweden.
SO Infection and immunity, (2006 Jun) Vol. 74, No. 6, pp. 3488-97.
Journal code: 0246127. ISSN: 0019-9567.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 200606
ED Entered STN: 23 May 2006
Last Updated on STN: 16 Jun 2006
Entered Medline: 15 Jun 2006

AB Bacterial adherence to mucosal surfaces is an important virulence trait of pathogenic bacteria. Adhesion of **enterotoxigenic Escherichia coli** (ETEC) to the intestine is mediated by a number of antigenically distinct colonization factors (CFs). One of the most common CFs is CFA/I. This has a fimbrial structure composed of a major repeating subunit, CfaB, and a single tip subunit, CfaE. The potential carbohydrate recognition by CFA/I was investigated by binding CFA/I-fimbriated bacteria and purified CFA/I fimbriae to a large number of variant glycosphingolipids separated on thin-layer chromatograms. For both fimbriated bacteria and purified fimbriae, specific interactions could be identified with a number of nonacid glycosphingolipids. These included glucosylceramide, lactosylceramide with phytosphingosine and/or hydroxy fatty acids, **neolactotetraosylceramide**, gangliotriaosylceramide, gangliotetraosylceramide, the H5 type 2 pentaglycosylceramide, the Lea-5 glycosphingolipid, the Lex-5 glycosphingolipid, and the Ley-6 glycosphingolipid. These glycosphingolipids were also recognized by recombinant *E. coli* expressing CFA/I in the absence of tip protein CfaE, as well as by purified fimbriae from the same strain. This demonstrates that the

glycosphingolipid-binding capacity of CFA/I resides in the major CfaB subunit.

L58 ANSWER 4 OF 16 MEDLINE on STN
AN 2004041592 MEDLINE
DN PubMed ID: 14576169
TI Carbohydrate recognition by enterohemorrhagic *Escherichia coli*: characterization of a novel glycosphingolipid from cat small intestine.
AU Teneberg Susann; Angstrom Jonas; Ljungh Asa
CS Institute of Medical Biochemistry, Goteborg University, SE 405 30 Goteborg, Sweden.. susann.teneberg@medkem.gu.se
SO Glycobiology, (2004 Feb) Vol. 14, No. 2, pp. 187-96. Electronic Publication: 2003-10-23.
Journal code: 9104124. ISSN: 0959-6658.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 200410
ED Entered STN: 27 Jan 2004
Last Updated on STN: 20 Oct 2004
Entered Medline: 19 Oct 2004
AB A key virulence trait of pathogenic bacteria is the ability to bind to receptors on mucosal cells. Here the potential glycosphingolipid receptors of enterohemorrhagic *Escherichia coli* were examined by binding of 35S-labeled bacteria to glycosphingolipids on thin-layer chromatograms. Thereby a selective interaction with two nonacid glycosphingolipids of cat small intestinal epithelium was found. The binding-active glycosphingolipids were isolated and, on the basis of mass spectrometry, proton NMR spectroscopy, and degradation studies, identified as Galalpha3Galbeta4Glcbeta1Cer (isoglobotriaosylceramide) and Galalpha3Galalpha3Galbeta4Glcbeta1Cer. The latter glycosphingolipid has not been described before. The interaction was not based on terminal Galalpha3 because the bacteria did not recognize the structurally related glycosphingolipids Galalpha3Galalpha4Galbeta4Glcbeta1Cer and Galalpha3Galbeta4GlcNAcbeta3Galbeta4Glcbeta1Cer (B5 glycosphingolipid). However, further binding assays using reference glycosphingolipids showed that the enterohemorrhagic *E. coli* also bound to lactosylceramide with phytosphingosine and/or hydroxy fatty acids, suggesting that the minimal structural element recognized is a correctly presented lactosyl unit. Further binding of neolactotetraosylceramide, lactotetraosylceramide, the Le(a)-5 glycosphingolipid, as well as a weak binding to gangliotriaosylceramide and gangliotetraosylceramide, was found in analogy with binding patterns that previously have been described for other bacteria classified as lactosylceramide-binding.

L58 ANSWER 5 OF 16 MEDLINE on STN
AN 2001698041 MEDLINE
DN PubMed ID: 11744628
TI Helicobacter pylori-binding gangliosides of human gastric adenocarcinoma.
AU Roche N; Larsson T; Angstrom J; Teneberg S
CS Institute of Medical Biochemistry, Goteborg University, P.O. Box 440, SE 405 30 Goteborg, Sweden.
SO Glycobiology, (2001 Nov) Vol. 11, No. 11, pp. 935-44.
Journal code: 9104124. ISSN: 0959-6658.
CY England: United Kingdom
DT (IN VITRO)

Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)

LA English

FS Priority Journals

EM 200203

ED Entered STN: 18 Dec 2001

Last Updated on STN: 26 Mar 2002

Entered Medline: 25 Mar 2002

AB Acidic and neutral glycosphingolipids were isolated from a human gastric adenocarcinoma, and binding of *Helicobacter pylori* to the isolated glycosphingolipids was assessed using the chromatogram binding assay. The isolated glycosphingolipids were characterized using fast atom bombardment mass spectrometry and by binding of antibodies and lectins. The predominating neutral glycosphingolipids were found to migrate in the di- to tetraglycosylceramide regions as revealed by anisaldehyde staining and detection with lectins. No binding of *H. pylori* to these compounds was obtained. The most abundant acidic glycosphingolipids, migrating as the GM3 ganglioside and sialyl-neolactotetraosylceramide, were not recognized by the bacteria. Instead, *H. pylori* selectively interacted with slow-migrating, low abundant gangliosides not detected by anisaldehyde staining. Binding-active gangliosides were isolated and characterized by mass spectrometry, proton nuclear magnetic resonance, and lectin binding as sialyl-neolactohexaosylceramide (NeuAcalpha3Galbeta4GlcNAcbeta3Galbeta4GlcNAcbeta3Galbeta4Glcbeta1Cer) and sialyl-neolactooctaosylceramide (NeuAcalpha3Galbeta4GlcNAcbeta3Galbeta4GlcNAcbeta3Galbeta4GlcNAcbeta3Galbeta4GlcNAcbeta3Galbeta4Glcbeta1Cer).

L58 ANSWER 6 OF 16 MEDLINE on STN

AN 2001111860 MEDLINE

DN PubMed ID: 11056399

TI Isolectins from *Solanum tuberosum* with different detailed carbohydrate binding specificities: unexpected recognition of lactosylceramide by N-acetyllactosamine-binding lectins.

AU Ciopraga J; **Angstrom J**; Bergstrom J; Larsson T; Karlsson N; Motas C; Gozia O; **Teneberg S**

CS Institute of Biochemistry, Romanian Academy, P.O. Box 78 200 Bucharest, 2, Romania.

SO Journal of biochemistry, (2000 Nov) Vol. 128, No. 5, pp. 855-67.

Journal code: 0376600. ISSN: 0021-924X.

CY Japan

DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)

LA English

FS Priority Journals

EM 200102

ED Entered STN: 22 Mar 2001

Last Updated on STN: 18 Dec 2002

Entered Medline: 8 Feb 2001

AB Glycosphingolipid recognition by two isolectins from *Solanum tuberosum* was compared by the chromatogram binding assay. One lectin (PL-I) was isolated from potato tubers by affinity chromatography, and identified by MALDI-TOF mass spectrometry as a homodimer with a subunit molecular mass of 63,000. The other (PL-II) was a commercial lectin, characterized as two homodimeric isolectins with subunit molecular masses of 52,000 and 55,000, respectively. Both lectins recognized N-acetyllactosamine-containing glycosphingolipids, but the fine details of their carbohydrate binding specificities differed. PL-II preferentially bound to glycosphingolipids with N-acetyllactosamine branches, as Galbeta4GlcNAcbeta6(Galbeta4GlcNAcbeta3)Galbeta4Glcbeta1Cer. PL-I also recognized this glycosphingolipid, but bound equally well to the linear

glycosphingolipid Galbeta4GlcNAcbeta3Galbeta4GlcNAcbeta3Galbeta4Glcbeta1Cer. **Neolactotetraosylceramide** and the B5 pentaglycosylceramide were also bound by PL-I, while other glycosphingolipids with only one N-acetyllactosamine unit were non-binding. Surprisingly, both lectins also bound to lactosylceramide, with an absolute requirement for sphingosine and non-hydroxy fatty acids. The inhibition of binding to both lactosylceramide and N-acetyllactosamine-containing glycosphingolipids by N-acetylchitotetraose suggests that lactosylceramide is also accommodated within the N-acetylchitotetraose/N-acetyllactosamine-binding sites of the lectins. Through docking of glycosphingolipids onto a three-dimensional model of the PL-I hevein binding domain, a Galbeta4GlcNAcbeta3Galbeta4 binding epitope was defined. Furthermore, direct involvement of the ceramide in the binding of lactosylceramide was suggested.

L58 ANSWER 7 OF 16 MEDLINE on STN
 AN 2001110159 MEDLINE
 DN PubMed ID: 11087709
 TI Inhibition of nonopsonic *Helicobacter pylori*-induced activation of human neutrophils by sialylated oligosaccharides.
 AU Teneberg S; Jurstrand M; Karlsson K A; Danielsson D
 CS Institute of Medical Biochemistry, Goteborg University, P.O. Box 440, SE 405 30 Goteborg, Sweden.
 SO Glycobiology, (2000 Nov) Vol. 10, No. 11, pp. 1171-81.
 Journal code: 9104124. ISSN: 0959-6658.
 CY ENGLAND: United Kingdom
 DT (IN VITRO)
 Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 LA English
 FS Priority Journals
 EM 200102
 ED Entered STN: 22 Mar 2001
 Last Updated on STN: 22 Mar 2001
 Entered Medline: 2 Feb 2001
 AB Certain strains of *Helicobacter pylori* have nonopsonic neutrophil-activating capacity. Some *H. pylori* strains and the neutrophil-activating protein of *H. pylori* (HPNAP) bind selectively to gangliosides of human neutrophils. To determine if there is a relationship between the neutrophil-activating capacity and the ganglioside-binding ability, a number of *H. pylori* strains, and HPNAP, were incubated with oligosaccharides, and the effects on the oxidative burst of subsequently challenged neutrophils was measured by chemiluminescence and flow cytometry. Both by chemiluminescence and flow cytometry a reduced response was obtained by incubation of *H. pylori* with sialic acid-terminated oligosaccharides, whereas lactose had no effect. The reductions obtained with different sialylated oligosaccharides varied to some extent between the *H. pylori* strains, but in general 3'-**sialyllactosamine** was the most efficient inhibitor. Challenge of neutrophils with HPNAP gave no response in the chemiluminescence assay, and a delayed moderate response with flow cytometry. Preincubation of the protein with 3'-**sialyllactosamine** gave a slight reduction of the response, while 3'-**sialyllactose** had no effect. The current results suggest that the nonopsonic *H. pylori*-induced activation of neutrophils occurs by lectinophagocytosis, the recognition of sialylated glycoconjugates on the neutrophil cell surface by a bacterial adhesin leads to phagocytosis and an oxidative burst with the production of reactive oxygen metabolites.

L58 ANSWER 8 OF 16 MEDLINE on STN

AN 2001060702 MEDLINE
 DN PubMed ID: 10965049
 TI Common architecture of the primary galactose binding sites of Erythrina corallodendron lectin and heat-labile **enterotoxin** from **Escherichia coli** in relation to the binding of branched **neolactohexaosylceramide**.
 AU **Teneberg S**; Berntsson A; **Angstrom J**
 CS Institute of Medical Biochemistry, Goteborg University, P.O. Box SE 405 30 Goteborg, Sweden.
 SO Journal of biochemistry, (2000 Sep) Vol. 128, No. 3, pp. 481-91. Journal code: 0376600. ISSN: 0021-924X.
 CY Japan
 DT (COMPARATIVE STUDY)
 Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 LA English
 FS Priority Journals
 EM 200012
 ED Entered STN: 22 Mar 2001
 Last Updated on STN: 18 Dec 2002
 Entered Medline: 22 Dec 2000
 AB The heat-labile **enterotoxin** from **Escherichia coli** (LT) is responsible for so-called traveller's **diarrhea** and is closely related to the cholera toxin (CT). Toxin binding to GM1 at the epithelial cell surface of the small intestine initiates the subsequent **diarrheal** disease. However, LT has a broader receptor specificity than CT in that it also binds to N-acetyllactosamine-terminated structures. The unrelated lectin from Erythrina corallodendron (ECorL) shares this latter binding property. The findings that both ECorL and porcine LT (pLT) bind to lactose as well as to **neolactotetraosylceramide** suggests a common structural theme in their respective primary binding sites. Superimposing the terminal galactose of the lactoses in the respective crystal structures of pLT and ECorL reveals striking structural similarities around the galactose despite the lack of sequence and folding homology, whereas the interactions of the penultimate GlcNAcb3 in the **neolactotetraosylceramide** differ. The binding of branched **neolactohexaosylceramide** to either protein reveals an enhanced affinity relative to **neolactotetraosylceramide**. The b3-linked branch is found to bind to the primary Gal binding pocket of both proteins, whereas the b6-linked branch outside this site provides additional interactions in accordance with the higher binding affinities found for this compound. While the remarkable architectural similarities of the primary galactose binding sites of pLT and ECorL point to a convergent evolution of these subsites, the distinguishing structural features determining the overall carbohydrate specificities are located in extended binding site regions. In pLT, Arg13 is thus found to play a crucial role in enhancing the affinity not only for N-acetyllactosamine-terminated structures but also for GM1 as compared to human LT (hLT) and CT. The physiological relevance of the binding of N-acetyllactosamine-containing glycoconjugates to LT and ECorL is briefly discussed.

L58 ANSWER 9 OF 16 MEDLINE on STN
 AN 1999054731 MEDLINE
 DN PubMed ID: 9832619
 TI Glycosphingolipid binding specificities of Neisseria meningitidis and Haemophilus influenzae: detection, isolation, and characterization of a binding-active glycosphingolipid from human oropharyngeal epithelium.
 AU Hugosson S; **Angstrom J**; Olsson B M; Bergstrom J; Fredlund H; Olcen P; **Teneberg S**

CS Department of Otorhinolaryngology, Orebro Medical Center Hospital, Orebro, Sweden.

SO Journal of biochemistry, (1998 Dec 1) Vol. 124, No. 6, pp. 1138-52.
Journal code: 0376600. ISSN: 0021-924X.

CY Japan

DT (COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)

LA English

FS Priority Journals

EM 199903

ED Entered STN: 24 Mar 1999
Last Updated on STN: 24 Mar 1999
Entered Medline: 9 Mar 1999

AB The glycosphingolipid binding specificities of Haemophilus influenzae and Neisseria meningitidis were investigated as to the binding of radiolabeled bacteria to glycosphingolipids on thin-layer chromatograms. Thereby, similar binding profiles, for the binding of the two bacteria to lactosylceramide, isoglobotriaosylceramide, gangliotriaosylceramide, gangliotetraosylceramide, lactotetraosylceramide, **neolactotetraosylceramide**, and sialylneolactoheptaosylceramide, were obtained. On a closer view the binding preferences of the bacteria could be differentiated into three groups. The first specificity is recognition of lactosylceramide. The second specificity is binding to gangliotriaosylceramide and gangliotetraosylceramide, since conversion of the acetamido group of the N-acetylgalactosamine of gangliotriaosylceramide and gangliotetraosylceramide to an amine prevented the binding of the bacteria, and thus the binding to these two glycosphingolipids represents a separate specificity from lactosylceramide recognition. Preincubation of H. influenzae with **neolactotetraose** inhibited the binding to **neolactotetraosylceramide**, while the binding to lactosylceramide, gangliotetraosylceramide, or lactotetraosylceramide was unaffected. Thus, the third binding specificity is represented by **neolactotetraosylceramide**, and involves recognition of other neolacto series glycosphingolipids with linear N-acetyllactosamine chains, such as sialylneolactoheptaosylceramide. The relevance of the detected binding specificities for adhesion to target cells was addressed as to the binding of the bacteria to glycosphingolipids from human granulocytes, epithelial cells of human nasopharyngeal tonsils and human plexus choroideus. Binding-active **neolactotetraosylceramide** was thereby detected in human granulocytes and the oropharyngeal epithelium.

L58 ANSWER 10 OF 16 MEDLINE on STN

AN 97323395 MEDLINE

DN PubMed ID: 9179843

TI Structural basis for differential receptor binding of cholera and **Escherichia coli** heat-labile toxins: influence of heterologous amino acid substitutions in the cholera B-subunit.

AU Backstrom M; Shahabi V; Johansson S; **Teneberg S**; Kjellberg A; **Miller-Podraza H**; Holmgren J; Lebens M

CS Department of Medical Microbiology and Immunology, Goteborg University, Sweden.

SO Molecular microbiology, (1997 May) Vol. 24, No. 3, pp. 489-97.
Journal code: 8712028. ISSN: 0950-382X.

CY ENGLAND: United Kingdom

DT (COMPARATIVE STUDY)
(IN VITRO)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)

LA English
FS Priority Journals
EM 199708
ED Entered STN: 25 Aug 1997
Last Updated on STN: 25 Aug 1997
Entered Medline: 13 Aug 1997
AB The closely related B-subunits of cholera toxin (CTB) and **Escherichia coli** heat-labile enterotoxin (LTB) both bind strongly to GM1 ganglioside receptors but LTB can also bind to additional glycolipids and glycoproteins. A number of mutant CTB subunits were generated by substituting CTB amino acids with those at the corresponding positions in LTB. These were used to investigate the influence of specific residues on receptor-binding specificity. A mutated CTB protein containing the first 25 residues of LTB in combination with LTB residues at positions 94 and 95, bound to the same extent as native LTB to both delipidized rabbit intestinal cell membranes, complex glycosphingolipids (polyglycosylceramides) and **neolactotetraosylceramide**, but not to non-GM1 intestinal glycosphingolipids. In contrast, when LTB amino acid substitutions in the 1-25 region were combined with those in the 75-83 region, a binding as strong as that of LTB to intestinal glycosphingolipids was observed. In addition, a mutant LTB with a single Gly-33-->Asp substitution that completely lacked affinity for both GM1 and non-GM1 glycosphingolipids could still bind to receptors in the intestinal cell membranes and to polyglycosylceramides. We conclude that the extra, non-GM1 receptors for LTB consist of both sialylated and non-sialylated glycoconjugates, and that the binding to either class of receptors is influenced by different amino acid residues within the protein.

L58 ANSWER 11 OF 16 MEDLINE on STN
AN 96375687 MEDLINE
DN PubMed ID: 8781976
TI Recognition of glycoconjugates by *Helicobacter pylori*: an apparently high-affinity binding of human polyglycosylceramides, a second sialic acid-based specificity.
AU **Miller-Podraza H**; Milh M A; Bergstrom J; **Karlsson K A**
CS Department of Medical Biochemistry, Goteborg University, Sweden.
SO Glycoconjugate journal, (1996 Jun) Vol. 13, No. 3, pp. 453-60.
Journal code: 8603310. ISSN: 0282-0080.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 199701
ED Entered STN: 28 Jan 1997
Last Updated on STN: 28 Jan 1997
Entered Medline: 7 Jan 1997
AB *Helicobacter pylori* has been reported to agglutinate erythrocytes and to bind to various other cells in a sialic acid-dependent way. The binding was inhibited by **sialyllactose** or fetuin and other sialylated glycoproteins. The specificity apparently requires bacterial growth on agar, since we found that it was lost after growth in the nutrient mixture Ham's F12. Instead, the bacteria bound with high affinity and in a sialic acid-dependent way to polyglycosylceramides of human erythrocytes, a still incompletely characterized group of complex glycolipids. Bacteria grown in F12 medium were metabolically labelled with 35S-methionine and analysed for binding to glycolipids on thin-layer chromatograms and to glycoproteins on blots after electrophoresis, with human erythrocyte glycoconjugates in focus. There was no binding to simpler gangliosides

including GM3 or sialylparagloboside, or to a mixture of brain gangliosides. In contrast, polyglycosylceramides of human erythrocyte membranes bound at a pmol level. The activity was eliminated by mild acid treatment, mild periodate oxidation or sialidase hydrolysis. Erythrocyte proteins as well as a range of reference glycoproteins did not bind except band 3, which was weakly active. However, this activity was resistant to periodate oxidation. These results indicate a second and novel sialic acid-recognizing specificity which is expressed independently of the previously described specificity.

L58 ANSWER 12 OF 16 MEDLINE on STN
AN 95151768 MEDLINE
DN PubMed ID: 7849044
TI Enhanced binding of **enterotoxigenic Escherichia coli** K99 to amide derivatives of the receptor ganglioside NeuGc-GM3.
AU Lanne B; Uggla L; Stenhagen G; Karlsson K A
CS Department of Medical Biochemistry, Goteborg University, Sweden.
SO Biochemistry, (1995 Feb 14) Vol. 34, No. 6, pp. 1845-50.
Journal code: 0370623. ISSN: 0006-2960.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 199503
ED Entered STN: 22 Mar 1995
Last Updated on STN: 22 Mar 1995
Entered Medline: 13 Mar 1995
AB A natural receptor in pig small intestine [Teneberg, S., Willemsen, P., de Graaf, F. K., & Karlsson, K.-A. (1990) FEBS Lett. 263, 10-14] for the **enterotoxigenic** bacteria **Escherichia coli** K99 is the ganglioside NeuGc-GM3 (NeuGc alpha 3Gal beta 4Glc beta Cer) [e.g., H. Smit, W. Gaastra, J. P. Kamerling, J. F. G. Vliegthart, & F. K. de Graaf (1984) Infect. Immun. 46, 578-584]. Chemical modifications of the carboxyl group of this ganglioside were performed, giving five different amides, the methyl ester, and the primary alcohol. The products were purified, and their structures were investigated by negative FAB mass spectrometry. Binding of *E. coli* K99 was tested by incubating 35S-labeled bacteria with derivatized compounds separated on thin-layer chromatograms. Modification of the carboxyl group to a primary amide strengthened the binding at least 5-fold, as estimated from autoradiography of dilutions on thin-layer plates. Some strengthening of the binding was also obtained with the methylamide as well as with the carboxyl group reduced to the alcohol. The ethylamide bound equally well as the underivatized NeuGc-GM3. Amide substituents as large as propyl amide and benzyl amide were still recognized by the bacteria, although they bound weaker. The methyl ester was not stable in the chromatogram-binding assay with silica gel and water present, and it reverted to the acid.

L58 ANSWER 13 OF 16 MEDLINE on STN
AN 95104315 MEDLINE
DN PubMed ID: 7528675
TI Alpha 2,3-sialyl and alpha 1,3-fucosyltransferase-dependent synthesis of sialyl Lewis x, an essential oligosaccharide present on L-selectin counterreceptors, in cultured endothelial cells.
AU Majuri M L; Pinola M; Niemela R; Tiisala S; **Natunen J**; Renkonen O; Renkonen R
CS Department of Bacteriology, University of Helsinki, Finland.

SO European journal of immunology, (1994 Dec) Vol. 24, No. 12, pp. 3205-10.
Journal code: 1273201. ISSN: 0014-2980.

CY GERMANY: Germany, Federal Republic of
DT (IN VITRO)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)

LA English
FS Priority Journals
EM 199501
ED Entered STN: 15 Feb 1995
Last Updated on STN: 6 Feb 1998
Entered Medline: 27 Jan 1995

AB Sialyl Lewis x (sLex) oligosaccharides have been shown to be present in counterreceptors for L-selectin. We and others have previously shown that high endothelial cells in lymph nodes and at sites of inflammation express sLex. Here we show that also cultured human umbilical vein endothelial cells (HUVEC) express sLex on their cell surface. This oligosaccharide is formed by sequential action of alpha 2,3-sialyl- (alpha 2,3-ST) and alpha 1,3-fucosyltransferases (alpha 1,3-FT) on N-acetyllactosamine. At least two of the several alpha 2,3-ST and four of the several alpha 1,3-FT are present in HUVEC. In functional assays both alpha 2,3-ST and alpha 1,3-FT activities were observed in HUVEC lysates with exogenous lactosamine and sialyllactosamine acceptors, leading to the generation of the sialyllactosamine and sLex sequences, respectively. TNF stimulation increased the level of mRNA expression of FT VI, and the alpha 1,3-FT activity in HUVEC. Taken together these data show that endothelial cells express sLex and that they possess mRNA as well as enzyme activities of several alpha 2,3-ST and alpha 1,3-FT necessary in the final steps of sLex synthesis. Furthermore, inflammatory cytokines such as TNF can enhance transferase activities relevant in generating putative L-selectin counterreceptors.

L58 ANSWER 14 OF 16 MEDLINE on STN
AN 94129215 MEDLINE
DN PubMed ID: 7507746
TI Monoclonal antibody against a lactose epitope of glycosphingolipids binds to melanoma tumour cells.

AU Ding K; Ekberg T; Zeuthen J; Teneberg S; Karlsson K A;
Rosen A
CS Department of Tumor Immunology, Wallenberg Laboratory, University of Lund, Sweden.

SO Glycoconjugate journal, (1993 Oct) Vol. 10, No. 5, pp. 395-405.
Journal code: 8603310. ISSN: 0282-0080.

CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)

LA English
FS Priority Journals
EM 199403
ED Entered STN: 18 Mar 1994
Last Updated on STN: 3 Feb 1997
Entered Medline: 8 Mar 1994

AB Mice were immunized with a neoglycoprotein consisting of a chemically modified carbohydrate moiety (reductively aminated 3'-sialyllactose) linked to human serum albumin. By this procedure an antibody response to the normally non-immunogenic carbohydrate structure was obtained. Hybridomas were established, and monoclonal antibodies were selected in ELISA based on their binding to the saccharide hapten, or to a lactosylceramide-mimicking neoglycolipid, lactose-bis-sulfone. One of the selected antibodies, 2H4, was of

particular interest, since it also bound to glycolipids present on melanoma cells. FACS analysis of a panel of 14 melanoma cell lines showed that the 2H4 antibody bound to the majority of these. In frozen, non-fixed sections or paraffin sections of biopsies the monoclonal antibody 2H4 stained melanoma cells, but not tumour infiltrating lymphocytes or normal skin. Detailed immunochemical analysis of 2H4, using thin layer chromatography revealed that it recognized an internal lactose epitope in several glycosphingolipids.

L58 ANSWER 15 OF 16 MEDLINE on STN
AN 86051591 MEDLINE
DN PubMed ID: 3840699
TI Mouse monoclonal antibodies with specificity for the melanoma-associated ganglioside **disialyllactosylceramide** (GD3) also react with the structural analogue disialylparagloboside.
AU Brodin T; Hellstrom I; Hellstrom K E; **Karlsson K A**; Sjogren H O; Stromberg N; Thurin J
SO Biochimica et biophysica acta, (1985 Dec 4) Vol. 837, No. 3, pp. 349-53. Journal code: 0217513. ISSN: 0006-3002.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 198601
ED Entered STN: 21 Mar 1990
Last Updated on STN: 21 Mar 1990
Entered Medline: 3 Jan 1986
AB A mouse monoclonal IgM antibody, 4.2, has previously been shown to bind preferentially to the surface of human malignant melanoma cells and to have specificity for the GD3 ganglioside (NeuAc alpha 2----8NeuAc alpha 2----3Gal beta 1----4GlcCer). Using overlay of antibodies on thin-layer chromatograms with glycolipids of various sources, it was shown that antibody 4.2, a further IgM and two IgG3 mouse monoclonal antibodies, selected on the basis of reactivity with GD3, also bound with similar strength to the structural analogue NeuAc alpha 2----8NeuAc alpha 2----3Gal beta 1----4GlcNac beta 1----3Gal beta 1----4GlcCer or disialylparagloboside. The SK-MEL 28 melanoma cell line used for immunization was shown to contain a large amount of GD3 but to lack disialylparagloboside. The demonstrated cross-reactivity may be of importance when considering the use of these antibodies for biochemical and medical purposes.

L58 ANSWER 16 OF 16 MEDLINE on STN
AN 80006650 MEDLINE
DN PubMed ID: 479198
TI Structural characterization of lactotetraosylceramide, a novel glycosphingolipid isolated from human meconium.
AU **Karlsson K A**; Larson G
SO The Journal of biological chemistry, (1979 Sep 25) Vol. 254, No. 18, pp. 9311-6. Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 197911
ED Entered STN: 15 Mar 1990
Last Updated on STN: 15 Mar 1990
Entered Medline: 21 Nov 1979

AB In the course of work on a systematic structural mapping of nonacid glycosphingolipids of human meconia, special attention was given to a major component preliminarily identified as an isomer of **neolactotetraosylceramide** (paragloboside). This component was isolated in its pure form from meconium of a blood group O individual and subjected to detailed structural analyses, using mass spectrometry and proton NMR spectroscopy on intact permethylated and permethylated-reduced (LiAlH₄) derivatives, and gas liquid chromatography on degradational products of native, permethylated, and permethylated-reduced derivatives. The isolated compound was conclusively shown to have the structure Galp beta 1 yields 3GlcNAcp beta 1 yields 3Galp beta 1 yields 4Glc beta 1 yields 1Cer, and is thus identified as lactotetraosylceramide. The major fatty acids were 2-hydroxy fatty acids with 16 and 20 to 24 carbon atoms, and the bases were sphingosine and phytosphingosine. This glycolipid, although not isolated and structurally characterized before, has long been thought of as a precursor substance of the Lewis active glycolipids and of ABH-active glycolipids with a type 1 saccharide chain.

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AN 2005:275685 BIOSIS

DN PREV200510055255

TI Recombinant probiotics for treatment and prevention of enterotoxigenic *Escherichia coli* diarrhea.

AU Paton, Adrienne W.; Jennings, Michael P.; Morona, Renato; Wang, Hui; Focareta, Antonio; Roddam, Louise F.; Paton, James C. [Reprint Author]

CS Univ Adelaide, Sch Mol and Biomed Sci, Adelaide, SA 5005, Australia
james.paton@adelaide.edu.au

SO Gastroenterology, (MAY 2005) Vol. 128, No. 5, pp. 1219-1228.

CODEN: GASTAB. ISSN: 0016-5085.

DT Article

LA English

ED Entered STN: 21 Jul 2005

Last Updated on STN: 21 Jul 2005

AB Background & Aims: We have developed a therapeutic strategy for gastrointestinal infections that is based on molecular mimicry of host receptors for bacterial toxins on the surface of harmless gut bacteria. The aim of this study was to apply this to the development of a recombinant probiotic for treatment and prevention of diarrheal disease caused by enterotoxigenic *Escherichia coli* strains that produce heat-labile enterotoxin. Methods: This was achieved by expressing glycosyltransferase genes from *Neisseria meningitidis* or *Campylobacter jejuni* in a harmless *Escherichia coli* strain (CWG:308), resulting in the production of a chimeric lipopolysaccharide capable of binding heat-labile

enterotoxin with high avidity. Results: The strongest heat-labile enterotoxin binding was achieved with a construct (CWG308:pLNT) that expresses a mimic of lacto-N-neotetraose, which neutralized $\geq 93.8\%$ of the heat-labile enterotoxin activity in culture lysates of diverse enterotoxigenic *Escherichia coli* strains of both human and porcine origin. When tested with purified heat-labile enterotoxin, it was capable of adsorbing approximately 5% of its own weight of toxin. Weaker toxin neutralization was achieved with a construct that mimicked the ganglioside GM2. Preabsorption with, or coadministration of, CWG308:pLNT also resulted in significant in vivo protection from heat-labile enterotoxin-induced fluid secretion in rabbit ligated ileal loops. Conclusions: Toxin-binding probiotics such as those described here have considerable potential for prophylaxis and treatment of enterotoxigenic *Escherichia coli*-induced travelers' diarrhea.

CC Pathology - Therapy 12512
 Digestive system - Physiology and biochemistry 14004
Digestive system - Pathology 14006
 Pharmacology - General 22002
 Physiology and biochemistry of bacteria 31000
 Medical and clinical microbiology - General and methods 36001

IT Major Concepts
 Pharmacology; Infection; Digestive System (Ingestion and Assimilation)

IT Diseases
 diarrhea: digestive system disease, symptom
 Diarrhea (MeSH)

IT Diseases
 gastrointestinal infection: digestive system disease, infectious disease

IT Chemicals & Biochemicals
 enterotoxin; lacto-N-neotetraose

ORGN Classifier
 Aerobic Helical or Vibrioid Gram-Negatives 06210
 Super Taxa
 Eubacteria; Bacteria; Microorganisms
 Organism Name
 Campylobacter jejuni (species)
 Taxa Notes
 Bacteria, Eubacteria, Microorganism

ORGN Classifier
 Enterobacteriaceae 06702
 Super Taxa
 Facultatively Anaerobic Gram-Negative Rods; Eubacteria; Bacteria; Microorganisms
 Organism Name
 Escherichia coli (species): strain-CWG308
 Taxa Notes
 Bacteria, Eubacteria, Microorganism

ORGN Classifier
 Leporidae 86040
 Super Taxa
 Lagomorpha; Mammalia; Vertebrata; Chordata; Animalia
 Organism Name
 rabbit (common)
 Taxa Notes
 Animals, Chordates, Lagomorphs, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Vertebrate

ORGN Classifier
 Neisseriaceae 06507
 Super Taxa
 Gram-Negative Aerobic Rods and Cocci; Eubacteria; Bacteria;

Microorganisms

Organism Name

Neisseria meningitidis (species)

Taxa Notes

Bacteria, Eubacteria, Microorganism

RN 13007-32-4 (lacto-N-neotetraose)

L80 ANSWER 2 OF 4 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

AN 2001:181018 BIOSIS

DN PREV200100181018

TI Nutritional formulations containing lacto-N-neotetraose.

AU Prieto, Pedro A. [Inventor]; Kroening, Terry A. [Inventor, Reprint author]

CS Gahanna, OH, USA

ASSIGNEE: Abbott Laboratories

PI US 6083934 20000704

SO Official Gazette of the United States Patent and Trademark Office Patents, (July 4, 2000) Vol. 1236, No. 1. e-file.

CODEN: OGUPE7. ISSN: 0098-1133.

DT Patent

LA English

ED Entered STN: 11 Apr 2001

Last Updated on STN: 18 Feb 2002

AB A nutritional formulation containing an effective amount of Lacto-N-neoTetraose to simulate the growth and/or metabolic activity of Bifidobacterium is provided. A process of inhibiting bacterial infections caused by Bacteroides, Clostridium, and E. coli including the step of feeding the nutritional composition to a subject is also provided.

NCL 514061000

CC General biology - Miscellaneous 00532

IT Major Concepts

Foods; Pediatrics (Human Medicine, Medical Sciences); Nutrition; Pharmacology

IT Diseases

bacterial infection: bacterial disease

Bacterial Infections (MeSH)

IT Chemicals & Biochemicals

lacto-N-neotetraose: feeding, nutrient, nutritional formulations

ORGN Classifier

Irregular Nonsporing Gram-Positive Rods 08890

Super Taxa

Actinomycetes and Related Organisms; Eubacteria; Bacteria; Microorganisms

Organism Name

Bifidobacterium: growth activity stimulation, metabolic activity stimulation

Taxa Notes

Bacteria, Eubacteria, Microorganisms

RN 13007-32-4 (lacto-N-neotetraose)

L80 ANSWER 3 OF 4 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

AN 1997:408381 BIOSIS

DN PREV199799714584

TI Immobilization of reducing sugars as toxin binding agents.

AU Nilsson, U. J.; Heerze, L. D.; Liu, Y.-C.; Armstrong, G. D.; Palcic, M. M.; Hindsgaul, O. [Reprint author]

CS Dep. Chem., Univ. Alberta, Edmonton, AB T6G 2G2, Canada

SO Bioconjugate Chemistry, (1997) Vol. 8, No. 4, pp. 466-471.

CODEN: BCCHE5. ISSN: 1043-1802.

DT Article

LA English
ED Entered STN: 24 Sep 1997
Last Updated on STN: 21 Nov 1997
AB A simple and economical procedure for the attachment of reducing sugars to aminated solid supports has been developed. Reaction of the amino groups on the solid support with p-nitrophenyl chloroformate, followed by 1,6-hexanediamine, yields a chain-extended amine to which reducing sugars can be attached while remaining accessible to macromolecules. Immobilization of the reducing sugars involves a simple incubation followed by trapping of the resulting glycosylamine with acetic anhydride and recovery of the unreacted sugar by filtration. This technique was used to immobilize lactose and sialyllactose onto silylaminated Chromosorb P, producing solid supports that effectively neutralized the activity of cholera toxin from *Vibrio cholerae* and heat-labile enterotoxin of enterotoxigenic *Escherichia coli*. The general applicability of such solid supports for toxin neutralization was further demonstrated by immobilization of the enzymatically synthesized alpha-Gal(1-3)beta-Gal(1-4)Glc trisaccharide, which produced a support that efficiently neutralized toxin A of *Clostridium difficile*. The results from this study suggest that these solid supports have the potential to serve as inexpensive therapeutics for bacterial toxin-mediated diarrheal diseases.
CC Biochemistry studies - General 10060
Biochemistry studies - Carbohydrates 10068
Digestive system - Pathology 14006
Toxicology - General and methods 22501
Physiology and biochemistry of bacteria 31000
Medical and clinical microbiology - Bacteriology 36002
IT Major Concepts
Biochemistry and Molecular Biophysics; Digestive System (Ingestion and Assimilation); Infection; Physiology; Toxicology
IT Chemicals & Biochemicals
P-NITROPHENYL CHLOROFORMATE; 1,6-HEXANEDIAMINE; SIALYLLACTOSE; LACTOSE
IT Miscellaneous Descriptors
BACTERIAL TOXINS; CHOLERA TOXIN; DIGESTIVE SYSTEM DISEASE; HOST; IMMOBILIZATION; LACTOSE; P-NITROPHENYL CHLOROFORMATE; PATHOGEN; REDUCING SUGAR; SIALYLLACTOSE; THERAPY PLANNING; TOXICITY; TOXICOLOGY; TOXIN A; TOXIN-MEDIATED DIARRHEA; 1,6-HEXANEDIAMINE
ORGN Classifier
Animalia 33000
Super Taxa
Animalia
Organism Name
animal
Animalia
Taxa Notes
Animals
ORGN Classifier
Endospore-forming Gram-Positives 07810
Super Taxa
Eubacteria; Bacteria; Microorganisms
Organism Name
endospore-forming gram-positive rods and cocci
Clostridium difficile
Taxa Notes
Bacteria, Eubacteria, Microorganisms
ORGN Classifier
Enterobacteriaceae 06702
Super Taxa
Facultatively Anaerobic Gram-Negative Rods; Eubacteria; Bacteria; Microorganisms

Organism Name
 Escherichia coli
 Taxa Notes
 Bacteria, Eubacteria, Microorganisms
 ORGN Classifier
 Vibrionaceae 06704
 Super Taxa
 Facultatively Anaerobic Gram-Negative Rods; Eubacteria; Bacteria;
 Microorganisms
 Organism Name
 Vibrio cholerae
 Taxa Notes
 Bacteria, Eubacteria, Microorganisms
 RN 7693-46-1 (P-NITROPHENYL CHLOROFORMATE)
 124-09-4 (1,6-HEXANEDIAMINE)
 3001-89-6Q (SIALYLLACTOSE)
 35890-38-1Q (SIALYLLACTOSE)
 63-42-3 (LACTOSE)
 11040-27-0Q (SIALYLLACTOSE)

 L80 ANSWER 4 OF 4 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
 AN 1995:300179 BIOSIS
 DN PREV199598314479
 TI Inhibition of cholera toxin by human milk fractions and sialyllactose.
 AU Idota, Tadashi; Kawakami, Hiroshi; Murakami, Yuji; Sugawara, Makihiro
 CS Technical Res. Inst., Snow Brand Milk Products Co. Ltd., 1-1-2 Minamidai,
 Kawagoe, Saitama 350-11, Japan
 SO Bioscience Biotechnology and Biochemistry, (1995) Vol. 59, No. 3, pp.
 417-419.
 ISSN: 0916-8451.
 DT Article
 LA English
 ED Entered STN: 11 Jul 1995
 Last Updated on STN: 11 Jul 1995
 AB The effects of human milk fractions on cholera toxin B subunit binding to
 monosialoganglioside 1 (G-M1) were investigated. Human milk, human
 defatted milk, whey, and a low-molecular-weight fraction of human milk
 inhibited the binding, but casein did not inhibit it. The inhibitory
 activity of whey from bovine-milk-based infant formula was less than that
 of whey from human milk. Differences in composition between human and
 bovine whey seemed to influence the extent of the inhibitory activity.
 Sialylated oligosaccharides were considered to be the possible components
 that inhibited cholera toxin. The effects of sialyllactose, a predominant
 sialylated component of human milk, on cholera toxin-induced
 diarrhea were investigated by the rabbit intestinal loop method.
 Sialyllactose inhibited the cholera toxin inducing fluid accumulation,
 although neither sialic acid nor lactose had an effect on it. The results
 suggest that sialyllactose is responsible for the inhibitory activity of
 milk on cholera toxin.
 CC Comparative biochemistry 10010
 Biochemistry methods - General 10050
 Biochemistry methods - Proteins, peptides and amino acids 10054
 Biochemistry methods - Carbohydrates 10058
 Biochemistry studies - General 10060
 Biochemistry studies - Proteins, peptides and amino acids 10064
 Biochemistry studies - Lipids 10066
 Biochemistry studies - Carbohydrates 10068
 Biophysics - Methods and techniques 10504
 Biophysics - Molecular properties and macromolecules 10506
 Physiology - General 12002

Physiology - Comparative 12003
 Pathology - General 12502
 Pathology - Comparative 12503
 Nutrition - General studies, nutritional status and methods 13202
 Nutrition - General dietary studies 13214
 Nutrition - Proteins, peptides and amino acids 13224
 Digestive system - Physiology and biochemistry 14004
 Digestive system - Pathology 14006
 Reproductive system - Physiology and biochemistry 16504
 Toxicology - General and methods 22501
 Toxicology - Antidotes and prevention 22505
 Physiology and biochemistry of bacteria 31000
 Medical and clinical microbiology - General and methods 36001
 Medical and clinical microbiology - Bacteriology 36002
 IT Major Concepts
 Biochemistry and Molecular Biophysics; Digestive System (Ingestion and Assimilation); Gastroenterology (Human Medicine, Medical Sciences); Infection; Methods and Techniques; Nutrition; Pathology; Physiology; Reproductive System (Reproduction); Toxicology
 IT Chemicals & Biochemicals
 SIALYLLACTOSE
 IT Miscellaneous Descriptors
 B SUBUNIT BINDING; BACTERIAL INFECTION; CASEIN; **DIARRHEA**;
 INHIBITORY ACTIVITY; LOW-MOLECULAR-WEIGHT FRACTION; RABBIT INTESTINAL LOOP METHOD; SIALYLATED OLIGOSACCHARIDES; WHEY
 ORGN Classifier
 Hominidae 86215
 Super Taxa
 Primates; Mammalia; Vertebrata; Chordata; Animalia
 Organism Name
 Hominidae
 Taxa Notes
 Animals, Chordates, Humans, Mammals, Primates, Vertebrates
 ORGN Classifier
 Leporidae 86040
 Super Taxa
 Lagomorpha; Mammalia; Vertebrata; Chordata; Animalia
 Organism Name
 Leporidae
 Taxa Notes
 Animals, Chordates, Lagomorphs, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Vertebrates
 ORGN Classifier
 Vibrionaceae 06704
 Super Taxa
 Facultatively Anaerobic Gram-Negative Rods; Eubacteria; Bacteria;
 Microorganisms
 Organism Name
 Vibrio cholerae
 Taxa Notes
 Bacteria, Eubacteria, Microorganisms
 RN 3001-89-6Q (SIALYLLACTOSE)
 35890-38-1Q (SIALYLLACTOSE)
 11040-27-0 (SIALYLLACTOSE)

=> => fil embase

FILE 'EMBASE' ENTERED AT 13:06:00 ON 07 JAN 2008

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FILE COVERS 1974 TO 7 Jan 2008 (20080107/ED)

EMBASE is now updated daily. SDI frequency remains weekly (default) and biweekly.

This file contains CAS Registry Numbers for easy and accurate substance identification.

Beginning January 2008, Elsevier will no longer provide EMTREE codes as part of the EMTREE thesaurus in EMBASE. Please update your current-awareness alerts (SDIs) if they contain EMTREE codes.

For further assistance, please contact your local helpdesk.

=> d all

L82 ANSWER 1 OF 1 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
AN 2002044256 EMBASE
TI Human milk oligosaccharides: A novel method provides insight into human genetics.
AU Erney R.; Hilty M.; Pickering L.; Ruiz-Palacios G.; Prieto P.
CS P. Prieto, Abbott Laboratories, Columbus, OH 43215, United States
SO Advances in Experimental Medicine and Biology, (2001) Vol. 501, pp. 285-297.
Refs: 12
ISSN: 0065-2598 CODEN: AEMBAP
CY United States
DT Journal; Conference Article; (Conference paper)
FS 022 Human Genetics
025 Hematology
029 Clinical and Experimental Biochemistry
LA English
SL English
ED Entered STN: 14 Feb 2002
Last Updated on STN: 14 Feb 2002
AB Human milk is a unique reservoir of oligosaccharides. The presence of many of these oligosaccharides is determined genetically and is related to the Lewis blood group and secretor antigen status of each donor. A method to quantitate neutral human milk oligosaccharides was developed. Sample preparation was based on a single centrifugation-filtration step that yields oligosaccharide extracts. These extracts first were fractionated to remove a significant portion of their lactose content and were analyzed using high-pH anion-exchange chromatography. Oligosaccharide profiles from 386 milk samples obtained in this fashion generated quantitative information on lactose, the neutral cores lacto-N-tetraose (LNT) and lacto-N-neotetraose (LNneoT), and the key fucosylated oligosaccharides. Additionally, the profiles provided genetic footprints of the Lewis and secretor status of the donors. Furthermore, unusual profiles that could not have been predicted from known genotypes were found. For this reason, milk glycoproteins were studied using carbohydrate-binding probes. Results confirm that oligosaccharides are an accurate predictor of the Lewis blood group status of the donor, and that glycosyltransferases have exquisite specificities. The data obtained in this study corroborate that Lewis-related antigens are tissue specific. This attribute of immunodominant carbohydrate sequences has significant implications for epidemiological studies of breast-fed infants.
CT Medical Descriptors:
accuracy

anion exchange chromatography
 antigenicity
 biochemical composition
 blood group Lewis system
 *breast milk
 carbohydrate analysis
 centrifugation
 conference paper
 extract
 filtration
 fractionation
 genotype
 human
 human genetics
 prediction
 priority journal
 protein determination
 technique

CT Drug Descriptors:

antigen: EC, endogenous compound
 carbohydrate: EC, endogenous compound
 glycoprotein: EC, endogenous compound
 glycosyltransferase: EC, endogenous compound
 lacto n neotetraose: EC, endogenous compound
 lacto n tetraose: EC, endogenous compound
 lactose: EC, endogenous compound
 *oligosaccharide: EC, endogenous compound
 unclassified drug

RN (glycosyltransferase) 9033-07-2; (lacto n neotetraose) 13007-32-4
 ; (lactose) 10039-26-6, 16984-38-6, 63-42-3, 64044-51-5

=> => d his

(FILE 'HOME' ENTERED AT 12:30:48 ON 07 JAN 2008)
 SET COST OFF

FILE 'REGISTRY' ENTERED AT 12:31:01 ON 07 JAN 2008

L1 1 S 35890-39-2
 L2 1 S 35890-38-1
 L3 1 S 13007-32-4

FILE 'MEDLINE' ENTERED AT 12:31:35 ON 07 JAN 2008

L4 0 S L1
 L5 277 S ?SIALYLLACTOS? OR ?SIALYLL LACTOS? OR ?SIALYLACTOS? OR ?SIALY
 L6 30 S ACETYL(S)NEURAMIN?(S)LACTOS?
 L7 179 S ACETYLLNEURAMIN?(S)LACTOS?
 L8 1 S NAN LACTOS?
 L9 425 S ?NEURAMINYLLACTOS? OR ?NEURAMIN? ?LACTOS?
 L10 1 S NAN LACTOS?
 L11 363 S ?NEURAMIN? ?LACTOS?
 L12 20 S LNNT
 L13 73 S NEOLACTOTETRAOS?
 L14 162 S LACTO (S) (NEOTETRAOS? OR NEO TETRAOS?)
 L15 1014 S L5-L14
 L16 6 S L15 AND (?DIARRH? OR ?DYSENTER? OR ?COLIC?)
 E DIARRHEA/CT
 E E3+ALL
 L17 35903 S E5+NT
 E E9+ALL

L18	1224	S E6	
		E DIARRHEA/CT	
		E E4+ALL	
		E E2+ALL	
L19	367	S E41	
		E DIARRHEA/CT	
		E E5+ALL	
		E E9+ALL	
L20	1577	S E9+NT	
		E DYSENTERY/CT	
		E E3+ALL	
L21	9964	S E9+NT	
		E E8+ALL	
L22	116171	S E4+NT	
		E COLIC/CT	
		E E3+ALL	
L23	2978	S E36	
L24	1	S L15 AND L17-L23	
L25	6	S L16, L24	
L26	58	S L15 AND (ESCHERICHIA OR "E") () COLI	
		E ESCHERICHIA/CT	
		E E4+ALL	
L27	43	S L15 AND E11+NT	
L28	9	S L15 AND E32+NT	
L29	44	S L15 AND E10+NT	
L30	59	S L26-L29	
		E ENTEROTOXIN/CT	
		E E5+ALL	
L31	19	S L15 AND E4+NT	
L32	71	S L25, L30, L31	
L33	12	S L15 AND ?ENTEROTOX?	
L34	71	S L32, L33	
		E GASTROINTESTINAL DISEASE/CT	
		E E4+ALL	
L35	30	S L15 AND E3+NT	
L36	98	S L34, L35	
L37	98	S L36 AND L1-L36	
L38	78	S L37 AND PY<=2002	
L39	20	S L37 NOT L38	
L40	1	S L39 AND 2005246876/AN	
L41	2	S L38 AND (79216779 OR 95252586)/AN	
L42	7	S L40, L41, L25 AND L1-L41	
L43	3	S L42 AND ?CERAMID?	
L44	7	S L42, L43	
		E ANGSTROM/AU	
L45	59	S E5, E6	
		E TENEBERG/AU	
L46	60	S E4, E5	
		E SAARINEN/AU	
L47	81	S E3, E14-E16, E18	
		E SATOMAA/AU	
L48	3	S E6	
		E ROCHE/AU	
L49	15	S E3	
		E ROCHE N/AU	
L50	116	S E3-E6, E13	
		E NATUNEN/AU	
L51	15	S E4, E5	
		E MILLER PODRAZA/AU	
L52	46	S E4, E5	

E MILLER H/AU
E PODRAZA/AU
L53 2 S E9
E KARLSSON/AU
E KARLSSON K/AU
L54 391 S E3,E4
L55 18 S E16-E18
E ABUL/AU
E ABUL M/AU
L56 12 S E7,E8
E ABULMILH/AU
L57 16 S L15 AND L45-L56
L58 16 S L57 AND L1-L57

FILE 'MEDLINE' ENTERED AT 12:57:53 ON 07 JAN 2008

FILE 'BIOSIS' ENTERED AT 12:58:22 ON 07 JAN 2008

FILE 'MEDLINE' ENTERED AT 12:58:33 ON 07 JAN 2008

L59 99 S L2 OR L3
L60 1 S L59 AND (?DIARRH? OR ?DYSENTER? OR ?COLIC?)
L61 0 S L59 AND L17-L23
L62 4 S L59 AND (ESCHERICHIA OR "E") ()COLI
L63 4 S L59 AND ESCHERICHIA+NT/CT
L64 1 S L59 AND ENTEROTOXINS+NT/CT
L65 3 S L59 AND GASTROINTESTINAL DISEASES+NT/CT
L66 8 S L60-L65
L67 7 S L66 NOT L44,L58

FILE 'BIOSIS' ENTERED AT 13:00:56 ON 07 JAN 2008

L68 13 S L1
L69 170 S L2
L70 48 S L3
L71 216 S L68-L70
L72 13 S L71 AND (ESCHERICHIA OR "E") ()COLI
L73 3 S L71 AND (?DIARRH? OR ?DYSENTER? OR ?COLIC?)
L74 2 S L71 AND ?ENTEROTOX?
L75 14 S L72-L74
L76 2 S L75 AND (2001:181018 OR 1995:300179)/AN
L77 9 S 14006/CC AND L71
L78 6 S L77 NOT L75
L79 3 S L77 NOT L78
L80 4 S L76,L79

FILE 'BIOSIS' ENTERED AT 13:04:07 ON 07 JAN 2008

FILE 'EMBASE' ENTERED AT 13:04:15 ON 07 JAN 2008

L81 25 S L1-L3
L82 1 S L81 AND 2002044256/AN

FILE 'EMBASE' ENTERED AT 13:06:00 ON 07 JAN 2008